REMARKS

The Office Action of April 22, 2008, has been received and reviewed. All claims currently under consideration stand rejected or objected to. Claim 1 is amended herein. New claims 27-30 are presented herein. Basis for new claims 27-30 can be found throughout the Specification and more specifically in FIG. 1 and Examples 1 and 3 of the Specification as filed. All amendments are made without prejudice or disclaimer. No new matter has been presented. Reconsideration is respectfully requested.

Claim Objections

Claim 1 stands objected to as "JAK-phosphatase" is misspelled therein. Applicants note that appropriate correction has been made.

Rejections Under 35 U.S.C. §§ 102(b) and 103(a)

Claims 1, 3, 11, 13, and 16 stand rejected under 35 U.S.C. §§ 102(b) and 103(a) as assertedly being anticipated by, or, in the alternative, rendered obvious over, Eyckerman et al. (1999. Eur Cytokine Netw. 10(4): 549-546) (hereinafter "Eyckerman"). Specifically, it was asserted that Eyckerman taught recombinant receptors comprising the mouse leptin receptor with at least one mutation in the cytoplasmic domain and a heterologous myc-tag. Office Action of April 22, 2008, at page 3. It was further asserted that the receptor of Eyckerman meets all the structural requirements of the claims, but does not meet the functional element of "wherein said recombinant receptor is activated by binding of a ligand to said extracellular ligand binding domain and by binding of a prey polypeptide to said heterologous bait polypeptide." *Id.* at page 4. However, the Examiner asserts that where the products seem identical except for the functional element, the burden shifts to the applicant. *Id.* Applicants respectfully traverse the rejections as hereinafter set forth.

Applicants note that a claim is only anticipated if each and every element as set forth in the claim is found, either expressly or inherently described, in a single prior art reference. *Verdegaal Bros. v. Union Oil Co. of Cal.*, 814 F.2d 628, 631 (Fed. Cir. 1987). Applicants respectfully assert that claims 1, 3, 11, 13, and 16 cannot be anticipated by Eyckerman as Eyckerman does not teach each and every element of the claims. For example, Eyckerman does

not teach a bait polypeptide.

The Examiner asserts, at page 4 of the Office Action of April 22, 2008, that the myc-tag present on the recombinant receptors of Eyckerman is equivalent to a heterologous bait polypeptide. Applicants respectfully disagree. For one of ordinary skill in the art, a clear distinction exists between a tag, such as a myc-tag, and a bait polypeptide. A tag is a short peptide of only a few amino acids, intended as a marker, whereas a bait polypeptide is a longer oligopeptide that forms a normal part of protein-protein interaction in a cellular system.

Further, Eyckerman does not disclose a prey/inhibitor fusion polypeptide as described in the instant specification. The prey, coupled to an inhibitor, is an essential element of the invention, as expressed in the claims. A person of ordinary skill in the art would not find such a prey fusion construct described or enabled in the teachings of Eyckerman. Further, one of ordinary skill in the art would have no knowledge (or motivation) that such a prey fusion construct could be used to inhibit the receptor. Additionally, even using the disclosure of the present application, it is unlikely that the Eyckerman receptor could be inhibited without burdensome experimentation in creating the anti-myc/inhibitor fusion necessary for myc-tagged protein of Eyckerman to be inhibited as claimed. Thus, no reasonable expectation of success exists in modifying the teachings of Eyckerman to arrive at the present claims.

The myc-tag is a short peptide of 10 amino acids that can be bound by an antibody, but is generally incapable of binding to a normal protein by the classical protein-protein interaction normally associated with cellular function; although the tag is extensively used in the art, there are no protein-protein interactions described with the myc-tag other than with myc-binding antibodies. Thus, inhibition of the myc-tagged protein of Eyckerman would only work through the cytoplasmic expression of a functional myc-binding antibody, fused to an inhibitor. It would have been clear to one of ordinary skill in the art that this would not work with classical heavy/light chain antibody complexes, as these are not found in the cytoplasm. If one of skill in the art attempted to develop a single chain antibody fused to an activation domain, such development would not yield predictable results, as the exact folding and requisite S-S bridge formation would be unpredictable for such a molecule without extensive experimentation. Moreover, even if one could obtain such a construct, it is unsure whether the inhibitor, fused to such a single chain antibody, could inhibit the recombinant receptor in coordination while the

anti-myc portion is bound to the myc-tag. Compared with bait/prey interactions, the anti-myc antibody interaction would be a bulky complex where steric hindrance would be expected to prevent inhibition of the receptor. Thus, applicants respectfully submit that the receptors of Eyckerman cannot be inherently capable of being inhibited when contacted with an antimyc/inhibitor fusion as the requisite anti-myc polypeptides have not been developed for intracellular use and there is no reasonable expectation of the successful function even if

In view of at least the foregoing, applicants respectfully request the withdrawal of the rejections of claims 1, 3, 11, 13, and 16 under 35 U.S.C. §§ 102(b) and 103(a) and reconsideration of same.

CONCLUSION

In light of the above amendments and remarks, applicants respectfully request reconsideration of the application. If questions remain after consideration of the foregoing, or if the Office should determine that there are additional issues which might be resolved by a telephone conference, the Office is kindly requested to contact applicants' attorney at the address or telephone number given herein.

Respectfully submitted,

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